

Biochemical Responses and Bioaccumulation Patterns in different Organs of *Cyprinus carpio* Exposed to Cadmium

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ABSTRACT

Background: Cadmium is a highly toxic heavy metal that poses serious threats to aquatic ecosystems by interfering with key biochemical and physiological functions in exposed organisms. The present study evaluated the tissue-specific accumulation of cadmium and its associated effects on enzymatic and metabolic profiles in *Cyprinus carpio* under chronic exposure conditions.

Methods: Healthy specimens of *Cyprinus carpio* (70–80 g) were acclimatized under laboratory conditions and divided into control (n = 5) and treated (n = 5) groups. The treated groups were exposed to cadmium chloride at concentrations of 2.0, 1.7, 1.4, and 0.2 mg/L for durations of 30, 40, 50, and 60 days, respectively. Serum biochemical markers including SGPT, SGOT, ALP, CPK, LDH, glucose, total protein, urea, calcium, and cholesterol were assessed. Cadmium bioaccumulation in liver, gills, intestine, and muscle tissues was quantified using atomic absorption spectrophotometry.

Results: Statistically significant ($P \leq 0.05$) and exposure duration–dependent variations were recorded across all evaluated parameters. Activities of SGPT, CPK, and LDH exhibited a consistent downward trend with increasing exposure time. In contrast, ALP and SGOT showed a biphasic response, characterized by an initial decline during 30–50 days of exposure followed by a marked elevation at 60 days, exceeding control values. Metabolic indicators such as glucose, total protein, calcium, and cholesterol were significantly reduced during early exposure phases, with partial recovery observed at prolonged exposure. Blood urea levels initially declined but increased sharply at later stages, suggesting progressive metabolic impairment. Cadmium distribution was clearly tissue-dependent, with the highest accumulation detected in the liver, followed by gills, intestine, and muscle. Hepatic cadmium levels approximately doubled after 60 days of exposure, indicating its central role in metal sequestration and detoxification.

Conclusion: Chronic exposure to cadmium induces pronounced biochemical and metabolic disruptions in *Cyprinus carpio*, along with significant tissue-specific accumulation patterns. These findings highlight the potential ecological risks associated with cadmium pollution and emphasize the need for continuous environmental monitoring and regulatory control.

INTRODUCTION

Heavy metals, especially cadmium (Cd), are well documented for their broad spectrum of toxic effects on fish and other aquatic organisms [43]. Cadmium is a non-essential element and exhibits pronounced toxicity when its concentration increases in aquatic systems. Likewise, non-essential metals such as mercury (Hg) and lead (Pb) serve no biological role and are highly hazardous to humans, even at very low exposure levels [20,31].

In humans, cigarette smoking is the primary source of cadmium exposure, but contaminated water, food, and air also contribute significantly. Acute cadmium poisoning may damage the testes, liver, and lungs, whereas chronic exposure is linked to renal failure, obstructive airway disease, emphysema, diabetic complications, hypertension, bone disorders, and immune suppression. These toxic effects result from cadmium's extremely slow excretion rate, with a biological half-life of approximately 15-20 years, leading to its accumulation in body tissues [1].

Due to intensified anthropogenic activities, cadmium has become one of the most dangerous heavy metals in aquatic ecosystems, threatening various fish species. Despite the well-known nutritional value of fish, environmental contamination especially by heavy metals has been widely reported. Heavy metals are among the most significant pollutants in aquatic ecosystems because of their toxicity and strong bioaccumulation potential in marine organisms such as fish [32].

Cadmium acts as a strong stressor for fish and can cause various pathophysiological changes, including reduced growth rates. Fish are capable of accumulating heavy metals in their tissues to concentrations higher than those found in the surrounding water through absorption across the gill surface and intestinal tract [26].

Fish are particularly sensitive to cadmium toxicity, which interferes with essential biochemical pathways and can result in severe physiological dysfunctions, reduced survival, or mortality. The accumulation of cadmium in edible fish tissues further represents a significant public health concern due to its potential entry into the human food chain.

Numerous studies have demonstrated that cadmium exposure via inhalation or ingestion can induce irreversible damage to critical organs, especially the kidneys, skeletal system, and respiratory tract. Such effects are most evident in populations with occupational or high environmental exposure, where cadmium levels are substantially elevated. Nevertheless, since the 1990s, epidemiological evidence—particularly from European studies—has suggested that even chronic low-level exposure in the general population may adversely affect renal function and bone integrity [32].

The aim of the present study was to investigate cadmium accumulation in the liver, intestine, gills, and muscles of *Cyprinus carpio* (Common carp) exposed to varying concentrations of the metal, and to evaluate associated biochemical changes in the blood.

METHODS

Sample collection

Healthy specimens of *Cyprinus carpio* were obtained from Sher Abad Hatchery, Mardan, and transported to the laboratory in aerated polyethylene bags to minimize transport stress. Upon arrival, the fish were acclimatized in 80 L glass aquaria under controlled laboratory conditions for 15 days to reduce handling stress and ensure adaptation to the experimental environment. Water quality parameters were regularly monitored throughout the acclimatization and experimental periods to maintain stable conditions.

Prior to stocking, aquaria were disinfected using potassium permanganate (KMnO₄) to prevent fungal, bacterial, and viral contamination. During the experimental period, fish were fed a commercial diet twice daily at 09:00 and 17:00 hours. A natural photoperiod (light–dark cycle) was maintained throughout the study. Approximately 40% of the aquarium water was replaced daily to ensure water quality stability. In addition, tanks were cleaned regularly to remove uneaten feed and fecal matter. Cadmium exposure was administered according to the designated concentrations, and treatments were consistently maintained across all

experimental groups throughout the exposure period.

Experimental Design

The experimental setup consisted of four treatment groups corresponding to exposure periods of 30, 40, 50, and 60 days, along with a control group. Each group included five fish of uniform size, with lengths ranging from 8–9 inches and body weights between 70–80 g. Cadmium chloride was administered at different concentrations according to exposure duration, i.e., 2.0 mg/L for 30 days, 1.7 mg/L for 40 days, 1.4 mg/L for 50 days, and 0.2 mg/L for 60 days. The control group was maintained under identical laboratory conditions without cadmium exposure and served as a baseline for comparison of biochemical and histological responses.

Blood Collection

The experimental design comprised four treatment groups corresponding to exposure durations of 30, 40, 50, and 60 days, along with a control group. Each group consisted of five fish of uniform size, measuring 8–9 inches in length and weighing 70–80 g. Cadmium chloride was administered at graded concentrations based on exposure duration, i.e., 2.0 mg/L for 30 days, 1.7 mg/L for 40 days, 1.4 mg/L for 50 days, and 0.2 mg/L for 60 days. The control group was maintained under identical environmental conditions without cadmium exposure and served as a reference for evaluating biochemical and histological alterations.

The biochemical parameters assessed included plasma protein, triglycerides, cholesterol, glucose, serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase (SGOT), creatine phosphokinase (CPK), lactate dehydrogenase (LDH), and alkaline phosphatase (ALP). Standardized protocols were followed for these assays: total protein was determined according to [30], cholesterol by the method of Henry and Henry (1974), glucose by [21], SGPT and SGOT by [39], CPK by [33], and LDH by [11].

Commercially available colorimetric assay kits were used, including SGOT (Elabscience, E-BC-K1301- M), CPK (Elabscience, E- BC- K900- M), LDH (Elabscience, E- BC- K771- M), ALP (Elabscience, E-BC-K009-M), SGPT (Elabscience, E-BC-F038), and glucose (GOD-POD method; Elabscience, E-BC-K234-S).

Tissue analysis for Bioaccumulation

For tissue-level analysis, approximately 1 g of liver, muscle, intestine, and gill tissues was excised from each specimen. The tissues were thoroughly rinsed with distilled water to eliminate external contaminants and processed separately for organ-specific assessment. The samples were then oven-dried at 80–90 °C until a constant weight was achieved. Subsequently, 1 g of each dried tissue sample was subjected to acid digestion following the procedures described by [42] and [13], with slight modifications as reported by [44].

The digestion procedure involved adding 10 mL of nitric acid (55%) and 5 mL of perchloric acid (70%) to each sample, followed by overnight incubation at room temperature. The next day, an additional 5 mL of nitric acid and 4 mL of perchloric acid were added. The samples were then heated on a hot plate at 200–250 °C until a clear solution was obtained. The appearance of dense white fumes indicated the completion of the digestion process.

After digestion, the samples were allowed to cool and then diluted with 10 mL of distilled water in the digestion flasks. The solutions were filtered through Whatman filter paper to remove any remaining particulates. The concentrations of cadmium in the tissue samples were determined using an atomic absorption spectrophotometer (Spectra AA-6300, Shimadzu, Japan).

RESULTS

The present study assessed cadmium bioaccumulation in different organs of *Cyprinus carpio* under varying exposure durations and evaluated its effects on blood biochemical parameters. Water quality parameters were regularly monitored throughout the experiment to maintain stable exposure conditions. The mean ($\pm$ SE) values recorded were: pH (7.10 ± 0.61), temperature (35 ± 0.37 °C), total hardness (87.27 ± 3.67 mg/L), dissolved oxygen (8.48 ± 0.56 mg/L), and total alkalinity (175.33 ± 2.91 mg/L) (Table 3). These parameters remained within

acceptable limits and were considered consistent across treatments, minimizing environmental interference.

Cadmium accumulation exhibited a clear tissue-dependent pattern (Tables: 2 Figures: 1), with the highest concentrations detected in the liver. Hepatic cadmium levels increased markedly from the control (0.01 ± 0.00) to 0.09 ± 0.02 after 30 days and further showed elevated accumulation by 60 days (0.02 ± 0.00 , Table 2), indicating strong uptake influenced by both exposure concentration and duration. Gill tissues also showed significant ($P \leq 0.05$) accumulation, reaching nearly 98% increase at 60 days (0.02 ± 0.00) compared to control (0.01 ± 0.00). The intestine exhibited a moderate but consistent increase, with levels rising to 0.38 ± 0.16 at 60 days from 0.08 ± 0.07 in controls. Muscle tissues similarly showed appreciable accumulation, with approximately a 79% increase after 60 days of exposure (Table 2).

Overall, cadmium distribution followed the order: liver > gills > muscle > intestine across all exposure periods. A comparison between short-term (30 days) and long-term (60 days) exposure confirmed a clear time-dependent increase in metal burden, highlighting cumulative uptake in response to prolonged exposure.

Biochemical analyses (Table 1; Figures: 2) revealed significant alterations ($P \leq 0.001$) in all measured enzymes. SGPT activity declined progressively from the control value (23.89 ± 1.15) to 16.00 ± 1.00 , 10.32 ± 1.00 , and 8.98 ± 1.00 at 30, 40, and 50 days, respectively. ALP activity initially decreased at 30 (45.23 ± 0.68), 40 (45.33 ± 0.68), and 50 days (40.33 ± 0.68), but increased significantly at 60 days (74.33 ± 0.88), exceeding the control value (68.23 ± 2.20). A similar biphasic response was observed for SGOT, which decreased during early exposure (10.62 ± 0.68 , 14.62 ± 0.68 , and 12.56 ± 0.68 at 30, 40, and 50 days, respectively) before rising at 60 days (23.67 ± 0.88), surpassing the control (18.23 ± 0.38).

CPK activity showed a steady decline from the control (590.21 ± 111.22) to 542.44 ± 0.58 , 510.44 ± 0.58 , 487.34 ± 0.58 , and 455.00 ± 0.44 with increasing exposure duration. Similarly, LDH activity decreased substantially from the control (987.12 ± 6.84) to 720.78 ± 0.77 and 730.45 ± 0.77 at 30 and 40 days, followed by further reduction at 50 and 60 days (710.00 ± 0.45).

Metabolic parameters also showed pronounced deviations (Table 1) Blood glucose levels dropped sharply from 103.33 ± 12.02 in the control group to 11.65 ± 0.78 , 8.45 ± 0.78 , and 6.32 ± 0.78 at 30, 40, and 50 days, respectively, with partial recovery at 60 days (23.67 ± 0.88). Total protein levels decreased from 12.37 ± 0.29 (control) to 8.78 ± 0.41 , 8.36 ± 0.41 , and 5.23 ± 0.41 during early exposure, followed by an increase to 9.25 ± 0.41 at 60 days. Blood urea levels declined initially (5.45 ± 0.22 , 4.55 ± 0.22 , and 4.23 ± 0.21) but increased sharply at 60 days (20.67 ± 0.44) compared to the control (12.78 ± 0.59). Calcium levels followed a similar decreasing trend during early exposure with slight recovery at later stages, while cholesterol levels also decreased initially with a modest increase at prolonged exposure.

Overall, these biochemical and physiological alterations were highly significant ($P \leq 0.001$), demonstrating that cadmium exposure severely disrupts metabolic homeostasis in *C. carpio*. The observed enzymatic disturbances likely reflect cadmium-induced organ toxicity, resulting from progressive accumulation in vital tissues and subsequent systemic distribution. Collectively, these findings emphasize the toxicological impact of cadmium on aquatic organisms and highlight the importance of monitoring heavy metal contamination in freshwater ecosystems.

Statistical Analysis

All data are presented as mean $\pm$ standard error of the mean (SEM). Differences in cadmium accumulation, enzymatic activities, and metabolite levels between control and treated groups were evaluated using one-way analysis of variance (ANOVA). Statistical analyses were performed using SPSS software. A probability value of $P \leq 0.05$ was considered statistically significant.

Figures

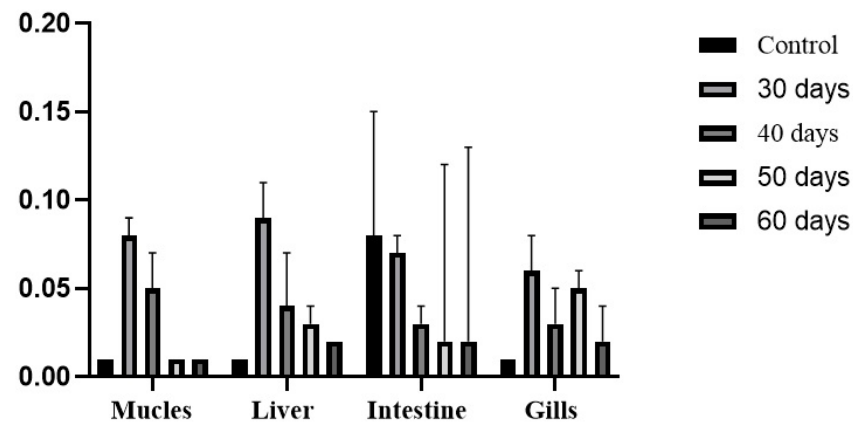


Figure 1: Tissue-specific cadmium bioaccumulation (mean ± SEM) in the muscles, liver, intestine, and gills of *Cyprinus carpio* under control conditions and following 30, 40, 50, and 60 days of exposure. Cadmium levels showed clear spatial and temporal variation, increasing progressively with exposure duration and accumulating most prominently in metabolically active tissues, particularly the liver and intestine. By comparison, lower concentrations were observed in the muscles and gills. Error bars indicate the standard error of the mean (SEM), representing variability among replicates.

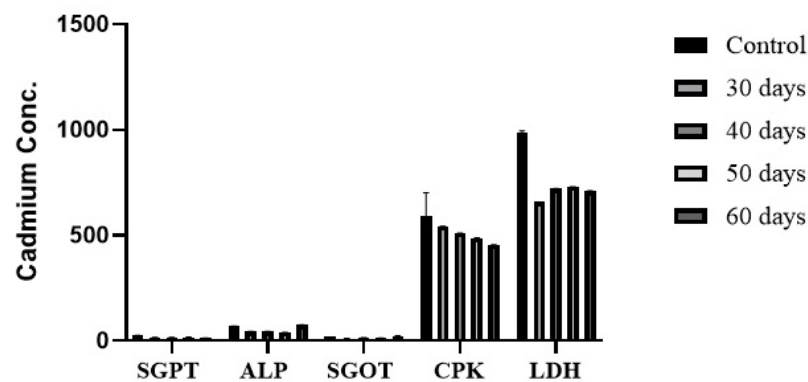


Figure 2: Serum activities of SGPT, ALP, SGOT, LDH, and CPK (mean ± SEM) were compared in *Cyprinus carpio* between control and cadmium-exposed groups at different exposure periods (n = 5 per group). A clear reduction in SGPT activity was observed in treated fish relative to the control, although some improvement was evident with longer exposure duration. Changes in the remaining enzymes indicate disruption of normal physiological functions under cadmium stress. Differences were considered statistically significant at $P \leq 0.05$.

Tables

Parameters	Control group Mean± SE (n=5)	Treated group (Exposure days and Cadmium concentrations)			
		Mean ± SE			
		30 days (n=5) Conc. 2 ml/L	40 days (n=5) Conc.1.7 ml/L	50 days (n=5) Conc.1.4 ml/L	60 days (n=5) Conc. 0.2 ml/L
SGPT (U/L)	23.89±1.15	16.00± 1.00*	10.32± 1.00*	8.98± 1.00*	17.67± 1.20
ALP (U/L)	68.23±2.20	45.23± 0.68**	45.33± 0.68**	40.33± 0.68**	74.33± 0.88
SGOT (U/L)	18.23±0.38	10.62± 0.68	14.62± 0.68*	12.56± 0.68*	23.67± 0.88
CPK (U/L)	590.21±111.22	542.44± 0.58	510.44± 0.58*	487.34± 0.58*	455.00± 0.44*
LDH (U/L)	987.12 ±6.84	657.68± 0.77*	720.78± 0.77*	730.45± 0.77*	710.00± 0.45
Glucose (mg/dL)	103.33± 12.02	11.65±0.78**	8.45±0.78*	6.32±0.78**	23.67±0.88
Protein (mg/dL)	12.37±0.29	8.78± 0.41***	8.36± 0.41**	5.23± 0.41*	9.25± 0.41*
Cholesterol (mg/dL)	43.81±5.98	8.57±0.26**	4.77±0.26*	4.22±0.26*	6.088±0.26
Urea (mg/dL)	12.78±0.59	5.45±0.22**	4.55±0.22*	4.23±0.21*	20.67±0.44
Calcium (mg/dL)	14.56±0.55	9.45± 0.34*	6.63± 0.34*	4.66± 0.34**	25.39± 0.34

Table 1: Mean ($\pm$ SEM) serum enzyme activities (SGPT, SGOT, ALP, CPK, and LDH) and metabolites (Glucose, Urea, protein, cholesterol and calcium) in *Cyprinus carpio* exposed to graded concentrations of cadmium chloride for 30, 40, 50, and 60 days. Values are presented in comparison with the control group ($n = 5$). Asterisks (*) indicate statistically significant differences relative to control ($P \leq 0.05$).

Tissues	Control group (Mean $\pm$ SEM) ($n=5$)	Treated group (Exposure to cadmium) (Mean $\pm$ SEM) (Percentage Difference)							
		30 days ($n=5$)		40 days ($n=5$)		50 days ($n=5$)		60 days ($n=5$)	
		Conc. (2ml/L)		Conc. (1.7ml/L)		Conc. (1.4ml/L)		Conc. (0.2ml/L)	
Liver	0.01 $\pm$ 0.00	0.09 $\pm$ 0.02	39 %	0.04 $\pm$ 0.03	49 %	0.03 $\pm$ 0.01	59%	0.02 $\pm$ 0.00	100 %
Muscles	0.01 $\pm$ 0.00	0.08 $\pm$ 0.01	70%	0.03 $\pm$ 0.01	75%	0.09 $\pm$ 0.05	78 %	0.01 $\pm$ 0.00	79%
Intestine	0.08 $\pm$ 0.07	0.28 $\pm$ 0.14	48 %	0.60 $\pm$ 0.18	52%	0.46 $\pm$ 0.11	57 %	0.38 $\pm$ 0.16	47 %
Gills	0.01 $\pm$ 0.00	0.06 $\pm$ 0.02	98 %	0.03 $\pm$ 0.02	97%	0.05 $\pm$ 0.01	98 %	0.02 $\pm$ 0.02	98%

Table 2: Tissue-specific accumulation of cadmium (mean $\pm$ SEM) and Percentage difference in liver, gills, intestine, and muscle of *Cyprinus carpio* after 30, 40, 50, and 60 days of exposure. Cadmium concentrations were determined using atomic absorption spectrophotometry. Values are expressed in mg/kg dry weight.

Temperature	Alkalinity	pH	Dissolve oxygen	Total hardness
35 $\pm$ 0.37	175.33 $\pm$ 2.91	7.10 $\pm$ 0.61	8.48 $\pm$ 0.56	87.27 $\pm$ 3.67

Table 3: Water quality parameters of experimental tanks recorded at different exposure intervals during the study period. Parameters include temperature ($^{\circ}$ C), pH, dissolved oxygen (mg/L), and other relevant physicochemical variables measured under control and cadmium-treated conditions. Values are expressed as mean $\pm$ SEM ($n = 5$). No significant variation in water quality parameters was observed across intervals, ensuring that experimental conditions remained stable throughout the exposure period.

DISCUSSION

The present study shows that cadmium accumulates in *Cyprinus carpio* in a clear tissue-specific and concentration-dependent pattern. Among the examined organs, the liver recorded the highest accumulation, followed by the gills, intestine, and muscles. Hepatic cadmium levels increased steadily with exposure duration, with varying percentage changes observed at each sampling point compared to the control group. This distribution pattern (liver > gills > intestine > muscles) reflects the physiological and functional roles of these tissues in detoxification, metabolism, and direct contact with the aquatic environment.

The higher accumulation in the liver can be explained by its central role in detoxification and its strong capacity for metal binding and sequestration. In fish exposed to heavy metals, the induction of metallothioneins (MTs) low-molecular-weight, cysteine-rich proteins is a well-recognized response. These proteins bind metal ions, regulate their intracellular concentration, and contribute significantly to detoxification and metal homeostasis [36].

The liver, being the primary organ for detoxification and metabolic regulation, exhibits a strong capacity for metal sequestration due to its high content of metallothioneins and other metal-binding proteins, which may explain the elevated cadmium accumulation observed [28]. It has been reported that heavy metals tend to accumulate in metabolically active tissues, with the liver showing a particularly high affinity for metal uptake and storage [45]. The present findings are consistent with this observation, as cadmium concentrations in the liver were higher than those recorded in the muscles and intestine. Similarly, previous studies have reported significant accumulation of heavy metals in hepatic tissues [24], while others have also demonstrated higher cadmium levels in liver compared to muscle tissue [25].

Gills also represent a major site of metal accumulation due to their direct contact with the surrounding aquatic environment and their role in ion regulation. The presence of chloride and other ion-transporting cells facilitates ion exchange, which may also enhance the uptake of dissolved metals. However, when metal concentrations exceed the physiological tolerance

threshold, toxic effects become evident [4,7].

The gills, being in direct contact with the surrounding aquatic environment, represent a primary site for metal uptake through respiratory exchange and ion transport, which explains their relatively high cadmium accumulation, particularly during early exposure periods [1]. As essential organs for respiration and osmoregulation, gills are highly susceptible to toxic insult. Cadmium-induced cellular damage can impair their physiological function by reducing the effective respiratory surface area and disrupting ion regulatory mechanisms [3]. Consistent with this, previous studies have also reported significant accumulation of cadmium in gill tissues [2].

The intestine showed moderate cadmium accumulation, which is likely associated with dietary uptake and ingestion of contaminated water. The present findings suggest that exposure duration significantly influences the degree of cadmium bioaccumulation in different tissues. [40] while studying heavy metal uptake in the intestine of *Mullus* spp., reported the following accumulation pattern: Zn > Cr > Ni > Cd. In a similar study, [45] documented considerable heavy metal deposition in the intestine of *Cyprinus carpio*, with an overall distribution pattern of liver > gills > intestine > muscles. The results of the present investigation are in agreement with these observations, as marked cadmium accumulation was also recorded in intestinal tissues.

Moreover, [44] reported significantly elevated cadmium levels in the intestine of crucian carp (*Carassius carassius*) exposed to waterborne cadmium. They also observed notable alterations in intestinal histoarchitecture, which are consistent with the morphological changes identified in the present study.

Muscle tissue exhibited the lowest cadmium accumulation, which may be attributed to its limited metal-binding capacity and relatively minor role in detoxification processes. Despite this, muscle is of particular importance in environmental monitoring studies due to its direct consumption by humans, making it a key indicator of food safety risk. Gills, which are continuously exposed to metal ions present in the surrounding water, also represent an important site of metal accumulation. From a public health perspective, fish skin is equally relevant, as it is often consumed together with muscle tissue and may therefore contribute to dietary metal exposure [46].

One study has already reported similar findings, observing no significant cadmium accumulation in fish muscle compared to other tissues and control groups [25]. Evidence further suggests that cadmium accumulation across different fish tissues, including muscle, may induce pathological alterations in vital organs such as the kidney and brain. The extent of cadmium bioaccumulation was influenced not only by tissue type but also by exposure duration. In the present study, cadmium levels in the liver and muscle increased with prolonged exposure, emphasizing the role of chronic exposure in metal accumulation dynamics.

Interestingly, gill cadmium concentrations were relatively higher during short-term exposure but declined over extended exposure periods. This pattern may be associated with redistribution of cadmium to internal organs, particularly the liver, or the activation of excretory and detoxification mechanisms over time. In contrast, the intestine showed comparatively stable cadmium levels, suggesting a degree of tissue-specific regulation and homeostatic control of metal uptake.

The observed alterations in enzymatic activities further substantiate the toxicological effects of cadmium exposure. Overall, enzyme responses were non-uniform and clearly time-dependent. SGPT, CPK, and LDH exhibited a consistent declining trend under exposure conditions, with CPK showing a moderate reduction and LDH demonstrating a more pronounced decrease. In contrast, ALP and SGOT displayed a biphasic response, characterized by an initial decline during the early exposure period (30–50 days), followed by a significant increase at 60 days. These findings indicate that enzymatic responses to cadmium are not uniformly suppressive but instead reflect a differential and adaptive pattern under prolonged exposure.

These changes point toward impaired liver function and overall metabolic imbalance in the exposed fish. Cadmium is known to affect enzyme systems through several mechanisms, including binding to sulfhydryl (-SH) groups, replacing essential metals such as zinc, and generating oxidative stress, all of which can suppress normal enzymatic activity. The decreased enzyme levels observed in this study therefore likely result from both the direct toxic action of

cadmium and secondary physiological stress caused by its accumulation in major metabolic organs.

Metal toxicity is well known to disrupt protein metabolism, which is often reflected in altered enzymatic profiles [29,48]. The reduction in plasma enzyme activities observed in the present study may be associated with changes in membrane permeability, leading to restricted leakage of enzymes into the bloodstream and their subsequent intracellular retention. Similar membrane damage induced by chromium exposure has been previously reported [14]. Additional mechanisms that may contribute to decreased enzyme activity include impaired intracellular enzyme synthesis and the presence of structurally altered or non-functional plasma enzymes that fail to perform their physiological roles (Sastry and Sunita, 1983). Moreover, increased urinary excretion of enzymes has also been suggested as a contributing factor to reduced serum enzyme levels [19]. The observed decline in GOT, SGPT, and LDH activities following cadmium exposure in the present study aligns with these previously reported findings. The changes in SGOT, SGPT, and LDH levels were statistically significant at $P \leq 0.01$.

[25] also supports the observed decrease in ALP activity following cadmium exposure. Similarly, [10] attributed the reduction in enzyme activity to the inhibitory effects of heavy metals on metabolic processes. In the present study, these changes were statistically significant ($P \leq 0.01$). The fluctuations in ALP levels reflect stress-induced alterations in enzymatic function. Comparable findings were reported by [12], who observed reduced ALP activity in *Labeo rohita* exposed to cypermethrin, thereby supporting the results of the current investigation. Furthermore, alterations in AST and ALT activities associated with stress-induced tissue damage have also been documented, reinforcing the present observations [17].

The present study revealed a significant reduction in serum CPK levels ($P \leq 0.05$). Similar observations were reported by [23], who also documented decreased CPK concentrations in fish exposed to cadmium. The reduced enzyme activity in blood serum may be attributed to the regenerative capacity of the liver, which limits enzyme leakage into circulation, or to the diversion of metabolic resources toward biosynthetic processes involved in tissue repair and recovery [6].

The observed reductions in glucose, blood urea, and calcium levels reflect heavy metal-induced physiological stress in fish. Similar hypoglycemic responses have been reported in *Cyprinus carpio* exposed to chromium [34], as well as in fish and mammals subjected to cadmium exposure [5,35], and in species such as *Heteropneustes fossilis*, *Labeo rohita*, and *Clarias gariepinus* under metal stress [42][38].

In contrast, the increase in urea levels observed in the present study is consistent with stress responses induced by heavy metals in aquatic organisms. Elevated blood urea nitrogen (BUN) and serum urea have previously been reported following exposure to cadmium and copper [32,41], indicating disrupted nitrogen metabolism and overall physiological stress.

The decline in serum cholesterol observed in the present study is consistent with previous reports documenting alterations in lipid profiles under heavy metal exposure [15,47,41,8]. Similarly, the reduction in serum total protein may indicate impaired hepatic or renal function [18,37,27]. This decrease may also be associated with enhanced protein catabolism or damage to erythrocytes induced by toxic stress conditions [10].

The present study provides clear evidence that chronic cadmium exposure induces pronounced bioaccumulation and significant biochemical disturbances in *Cyprinus carpio*. Cadmium accumulation followed a distinct tissue-specific and exposure duration-dependent pattern, with the highest concentrations detected in the liver, followed by the gills, intestine, and muscles. This distribution is consistent with the physiological roles of these organs, particularly the liver, which plays a central role in detoxification and metal sequestration. The observed reductions in key enzymatic activities and metabolic parameters reflect substantial physiological stress and cellular dysfunction, indicating impairment of vital organ systems, especially the liver and kidneys. Collectively, these changes are characteristic of cadmium-induced toxicity and disruption of normal metabolic homeostasis.

Overall, the findings highlight the deleterious effects of cadmium contamination on aquatic organisms and underscore its potential ecological and public health risks. The biochemical and

enzymatic alterations observed in the present study may serve as sensitive and reliable biomarkers for assessing heavy metal exposure in aquatic environments. Consequently, continuous environmental monitoring and strict regulatory control are necessary to reduce cadmium pollution and safeguard aquatic biodiversity as well as food chain safety.

CONFLICT OF INTEREST

There is no conflict of interest disclosed by the authors in the publication of this manuscript.

AUTHOR CONTRIBUTIONS

Dr. Ali Muhammad Yousafzai contributed to the conception and design of the study, supervised the experimental work, and provided critical guidance throughout data collection, analysis, and interpretation. The author also contributed to the drafting and critical revision of the manuscript and approved the final version for submission.

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Declaration of Generative AI and AI-assisted Technologies in the Writing Process

During the revision of this work, the authors used multiple AI tools to improve language and readability. After using these tools, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

ETHICS STATEMENT

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